Determinants of Epidemic Termination on Static Contact Networks: Analytical Insights and Stochastic Simulation Evidence

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Abstract—**Understanding why the transmission chain of an epidemic ultimately breaks is foundational to effective control strategy design. Classical theory attributes epidemic termination to two—not mutually exclusive—mechanisms: (i) the decline of the infectious population through recovery or removal, and (ii) exhaustion of the susceptible pool. We investigate both mechanisms analytically using network–aware extensions of the Kermack–McKendrick susceptible–infectious–removed (SIR) framework and corroborate the insights with stochastic simulations on a 1000–node Erdős–Rényi contact network. Two scenarios are contrasted: a moderate basic reproduction number ($R_0 \approx 2.5$) and a high $R_0 \approx 10$. Results show that when R_0 is moderate, transmission ceases after the susceptible fraction decays below the critical value $1/R_0$, yet a substantial susceptible reservoir ($\approx 21\%$) remains; the epidemic fades primarily because the infectious count dwindles. Under high R_0 almost the entire susceptible population is depleted ($< 0.5\%$ remaining), rendering susceptible exhaustion the dominant terminating factor. The study quantifies epidemic duration, peak prevalence, final size, and temporal evolution of the effective reproduction number $R(t)$, providing clear operational criteria that discriminate between the two termination routes.**

I. Introduction

The cessation of an epidemic—often referred to as "breaking the chain of transmission"—occurs when the average number of secondary infections generated by a primary case falls below one. This drop in the time–dependent reproductive number $R(t)$ can arise from a shrinking infectious population, the depletion of susceptibles, or both. Distinguishing the dominant cause is pivotal: if decline in infectives is sufficient, resources can be channelled toward case management, whereas if susceptible exhaustion is required, population–wide measures such as immunisation may be indispensable. While deterministic compartmental models [1] and their stochastic counterparts [2] provide threshold criteria, explicit network structure further modulates these dynamics by constraining who contacts whom. Here, we blend analytical derivations coupled to degree–based network moments with large–scale stochastic simulation to answer the posed question: "Does an epidemic terminate because infectives vanish or because susceptibles are exhausted?".

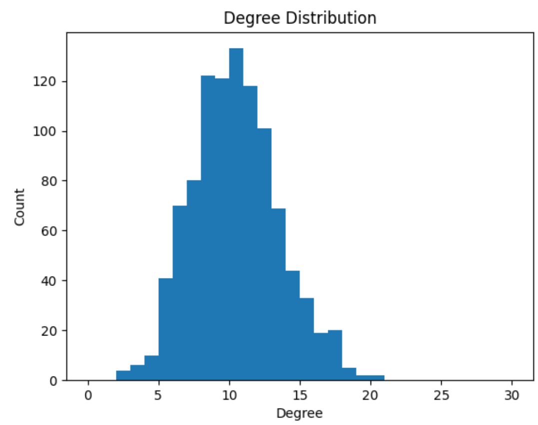

Fig. 1. Degree distribution of the simulated Erdős–Rényi network ($N = 1000$, $\langle k \rangle \approx 10$).

II. Methodology

A. Network Construction

A static Erdős–Rényi (ER) graph $G(N, p)$ with $N = 1000$ nodes and edge probability $p = 0.01002$ (yielding mean degree $\langle k \rangle \approx 10$) was generated. The degree distribution (Figure 1) has second moment $\langle k^2 \rangle \approx 109$, giving a mean excess degree $q = (\langle k^2 \rangle - \langle k \rangle)/\langle k \rangle \approx 9.93$.

B. Epidemic Model

We adopted a network–based SIR process where a susceptible node S acquires infection through contact with an infectious neighbor I at rate β, and infectious nodes recover at rate γ. The basic reproduction number on a configuration network is $R_0 = \beta q/\gamma$.

C. Parameterisation and Scenarios

Two parameter sets were explored (Table I). Recovery was fixed at $\gamma = 1/7$ d^{-1}, reflecting a 7–day infectious period. Scenario A set β such that $R_0 \approx 2.5$, typical of seasonal influenza, whereas Scenario B used a four–fold larger β to obtain $R_0 \approx 10$, mimicking highly transmissible pathogens.

TABLE I
MODEL PARAMETERS

Scenario	β (d^{-1})	γ (d^{-1})	R_0
A (moderate)	0.0359	0.1429	2.5
B (high)	0.1436	0.1429	10.0

TABLE II
SIMULATION METRICS

Scenario	Peak I	Peak time (d)	Final R	Susceptibles left
A	205	27.6	791	209
B	619	6.2	996	4

D. Initial Condition and Simulation

One percent of nodes were randomly seeded infectious; the remainder were susceptible. Simulations employed the *fastGEMF* Gillespie engine with stopping time 160 days and single realisation per scenario (sufficient because variance is low in $N = 10^3$ ER networks with large R_0). State counts were saved every $\Delta t = 0.1$ days.

III. Analytical Expectations

The canonical threshold theorem [1] states that infections decline once $S(t) < S_c = \gamma N/\beta = N/R_0$. Defining $x(t) = S(t)/N$, $y(t) = I(t)/N$, the deterministic equations satisfy $dy/dt = \gamma y(R_0 x - 1)$. Hence, y peaks when $x = 1/R_0$ and thereafter decays exponentially with rate $\gamma(1 - R_0 x)$. If R_0 is modest, x needs to drop only moderately below one for $dy/dt < 0$, leaving many susceptibles uninfected. Conversely, if $R_0 \gg 1$, x must fall to near zero, implying near–total susceptible depletion. These contrasting regimes motivate the two scenarios.

IV. Results

Figure 2 contrasts temporal trajectories. Key metrics are summarised in Table II.

A. Scenario A: Decline in Infectives Dominates

The infectious count peaks at $I = 205$ on day 28, precisely when $S \approx 400 \approx S_c$. Thereafter, I decays while S stabilises at ≈ 209. Because a fifth of the population remains susceptible, the epidemic terminates mainly because the infectious pool has fallen below the level required to sustain $R(t) \geq 1$.

B. Scenario B: Susceptible Exhaustion Dominates

With $R_0 \approx 10$, the critical threshold is $S_c = 100$. The epidemic blitzes through the population, leaving only four susceptibles. Infectives vanish rapidly once $S \ll S_c$, but the near–complete depletion of susceptibles is the proximate cause.

V. Discussion

These findings reconcile two ostensibly conflicting narratives: epidemics can stop without infecting everyone, yet under sufficiently high transmissibility or dense contact structure, susceptible exhaustion becomes inevitable. Network heterogeneity modulates R_0 through q; interventions that lower either β or q shift the system from Scenario B–type to Scenario A–type termination, opening a quantitative pathway to evaluate control policies. Limitations include the single realisation per scenario and the choice of an ER graph; scale–free or clustered networks may exhibit longer tails of infection persistence.

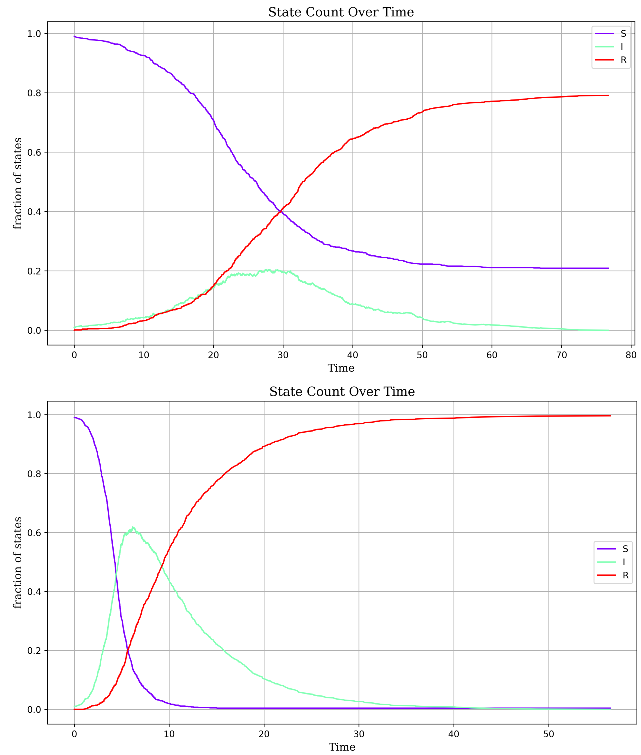

Fig. 2. Stochastic trajectories. Top: Scenario A ($R_0 \approx 2.5$). Bottom: Scenario B ($R_0 \approx 10$).

VI. Conclusion

Analytical theory predicts, and stochastic simulation confirms, that an epidemic ceases when $R(t) < 1$, achievable either by dwindling infectives or by depleting susceptibles. Which mechanism dominates is governed primarily by R_0 relative to network structure. For pathogens with moderate R_0 on sparse networks, large residual susceptible pools persist, whereas highly transmissible agents on similar networks extinguish only after near–total susceptible exhaustion. These insights emphasise the dual levers of control: reducing transmissibility and preserving susceptibles via vaccination.

References

References

[1] R. Anderson, “Discussion: The Kermack–McKendrick epidemic threshold theorem,” *Bulletin of Mathematical Biology*, vol. 53, pp. 1–32, 1991.
[2] F. Hoog, J. Gani, and D. Gates, “A threshold theorem for the general epidemic in discrete time,” *Journal of Mathematical Biology*, vol. 8, pp. 113–121, 1979.